do VEPs.[1] However, the comparison of VEP and behavioral data on the same individual in Fig. 2 (N. B. at 7 to 8 weeks) indicates quite close agreement, as does the comparison of the two 3-week-old infants' VEP with the 1-month group behavioral data; VEP and behavioral measures on a single 6-month-old individual[5]; and the individual VEP-based CSFs reported by Pirchio et al.[6] in comparison with behaviorally determined CSFs at 2 to 3 months.[2, 3] Comparison of data between different experiments must also consider the effects of stimulus variables. In the present study, behavioral data were obtained with a stimulus luminance 0.5 log units higher than that used for the VEP testing; on those grounds, the VEP results might be relative underestimates of performance. Further studies employing VEP and behavioral measures on the same infants by the same stimuli would be desirable to confirm the relationship between the two measures.

Insofar as data from the different methods may be compared, the VEP data from the two 3-week-old infants shown in Fig. 1 and the mean behavioral data for 5-week-olds show only modest improvements in acuity and contrast sensitivity over our neonatal VEP data. A larger increase in sensitivity is shown behaviorally between 1 and 2 months. If these comparisons are valid, they suggest that visual maturation may accelerate after the first month of life.

In interpreting VEP data, it should be borne in mind that each study uses a particular rate of temporal modulation of the stimulus. Little is known of the temporal properties of the infant visual system, and it is possible that use of different temporal parameters might lead to different estimates of visual spatial performance.

The acuity and sensitivity reported here for neonates may appear extremely poor in comparison to adult vision. However, they are adequate to provide useful visual information, especially over the short distances where most stimuli that are significant for the infant appear. For example, an acuity of 0.8 c/deg will preserve the over-all shape of the mother's face and its principal features at distances of 50 cm or less.

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REFERENCES

1. Dobson, V., and Teller, D. Y.: Visual acuity in human infants: a review and comparison of behavioral and electrophysiological studies, Vision Res. **18**:1469, 1978.
2. Atkinson, J., Braddick, O., and Moar, K.: Development of contrast sensitivity over the first three months of life in the human infant, Vision Res. **17**:1037, 1977.
3. Banks, M. S., and Salapatek, P.: Acuity and contrast sensitivity in 1-, 2-, and 3-month-old human infants, INVEST. OPHTHALMOL. VISUAL SCI. **17**:361, 1978.
4. Marg, E., Freeman, D. N., Peltzman, P. and Goldstein, P. J.: Visual acuity development in human infants: evoked potential measurements, INVEST. OPHTHALMOL. **15**:150, 1976.
5. Harris, L., Atkinson, J., and Braddick, O.: Visual contrast sensitivity of a 6 month-old infant measured by the evoked potential, Nature **264**:570, 1976.
6. Pirchio, M., Spinelli, D., Fiorentini, A., and Maffei, L.: Infant contrast sensitivity evaluated by evoked potentials. Brain Res. **141**:179, 1978.
7. Harter, M. R. Deaton, F. R., and Odom, J. V.: Visual evoked potentials to checkerboard flashes in infants from six days to six months. *In* Desmedt, J., editor: New Developments in Visual Evoked Potentials of the Human Brain. Oxford, 1977, Oxford University Press.
8. Regan, D.: Recent advances in electrical recording from the human brain, Nature **253**:401, 1975.
9. Campbell, F. W., and Maffei, L.: Electrophysiological evidence for the existence of orientation and size detectors in the human visual system J. Physiol. (Lond.) **207**:635, 1970.
10. Gorman, J. J., Cogan, D. G., and Gellis, S. S.: An apparatus for grading the visual acuity on the basis of optokinetic nystagmus, Pediatrics **19**:1088, 1957.
11. Dayton, G. D., Jones, M. H., Aiu, P., Rawson, R. A., Steele, B., and Rose, M.: Developmental study of co-ordinated eye movements in the human infant, Arch. Ophthalmol. **71**:865, 1964.

Different rates of functional recovery of eye movements during orthoptics treatment in an adult amblyope.

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Although it is common clinical knowledge that oculomotor control appears to normalize during the course

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of successful orthoptics therapy for amblyopia, reports providing a quantitative analysis of eye movements during extended periods of treatment are lacking. We provide for the first time such a report in an adult amblyope. Aspects of eye movement control that tended to normalize with therapy include drift amplitude and velocity, duration and frequency of steady fixation, and pursuit gain. These results suggest that smooth pursuit control can be modified, even in an adult amblyope. Aspects of eye movement control that remained abnormal throughout therapy, in spite of normalization of visual acuity and centralization of fixation, include increased saccadic latencies, use of large saccades during small-amplitude pursuit tracking, and static overshooting. These results suggest that certain aspects of saccadic and pursuit control could either no longer be modified or would require longer periods for this to occur.

Orthoptics is a nonsurgical method of developing comfortable, binocular vision in patients who have vision anomalies such as amblyopia[1] and strabismus[1, 2] which may impede sensory and motor fusion. It is common clinical knowledge that oculomotor control appears to normalize during the course of successful orthoptics therapy for amblyopia. Although reports are sorely needed which provide a quantitative analysis of changes in various aspects of oculomotor control obtained by objective eye movement recording performed *concurrent* with treatment,[1, 3] they are not evident in the literature. However, von Noorden and Burian[4] provided ac-electro-oculographic recordings of fixation and saccadic movements in one young amblyope before and after direct occlusion therapy and found eye movements to be normal once amblyopia was corrected. However, this information (only a small part of a larger study on fixation in amblyopia) was presented in a nonquantitative manner; furthermore, eye movements were not recorded *during* the course of treatment. Thus careful quantitative analysis of those aspects of oculomotor control that changed during amblyopia treatment, as well as their rate of change, are unknown. We provide for the first time such a report.

In our study, the fixational, saccadic, and pursuit eye movements of one adult amblyope were recorded on several occasions over an 8-month period of successful orthoptics treatment for amblyopia.

Methods. A photoelectric method was used to record horizontal eye position.[5] With this method, the amount of infrared light reflected from the horizontal limbal regions was monitored, and this was linear for the range of movements recorded. The bandwidth of the entire recording system was 75 Hz (−3 dB). Resolution was approximately 12 min arc. A chinrest and headrest, as well as a bite bar covered with dental impression material, were used to stabilize the head. Spectacle correction was worn throughout test periods.

A PDP8/I minicomputer was used to move a small spot of light (3.5 to 6.0 min arc) horizontally on a display monitor placed either 57 or 91 cm away on the subject's midline. Target luminance was maintained at least 1 log unit above screen luminance. Fixation was tested by having the subject maintain gaze on (for 15 to 120 sec) the target which was placed either on the midline or 2.5 or 5.0 deg to the left or right of the midline. Drift amplitude and velocity during fixation were determined at four test sessions; at each session, the same 15 to 30 sec portion of record was used to obtain all drift measures. Drift amplitude was determined in two ways. (1) The maximum peak-to-peak drift amplitude, regardless of time required for completion of the movement, was found, and (2) maximum peak-to-peak drift amplitude during consecutive 1 sec fixation intervals was obtained and averaged. Drift velocity was also determined in two ways. (1) Maximum drift velocity, regardless of time required for completion of the movement, was obtained, and (2) maximum drift velocity during a 200 msec period for consecutive 1 sec intervals was obtained and averaged. Saccadic movements were tested by having the subject track the spot moving either in random horizontal step displacements ranging from 0.25 to 8.5 deg in amplitude or in predictable steps having a frequency of 0.5 Hz with amplitudes of 0.6, 1.25, 2.5, 5.0, or 10.00 deg. Pulse inputs were also used. Pursuit was tested by having the subject follow the target moving within a range of constant velocities (0.95 to 6.75 deg/sec) and fixed amplitudes (1, 2, 4, or 8 deg) in several possible combinations. Mean smooth-pursuit gain was determined by averaging values of smooth-pursuit gain (eye velocity divided by target velocity) for each individual ramp segment of smooth tracking for 5 to 15 cycles of target motion.

Case history. The patient was an 18-year-old white man who came to our clinic to obtain a replacement for his spectacles which had been lost 1 year earlier. He reported that vision had always been poor in the left eye and that due to a birth trauma, his left eye had been totally occluded during the first 2 days of life. Refraction was +5.00 diopters in the left eye (20/230) and +3.00 diopters in the right eye (20/15). Although the initial cover test indicated 3 prism diopters of left eso-

tropia with 2 prism diopters of left hypertropia, these findings were confounded by unsteady fixation in the amblyopic eye. On later occasions as fixation became steadier, subsequent repeated cover tests clearly showed the absence of any strabismus. Initial visuoscopy indicated 10 prism diopters of temporal and 5 prism diopters of superior unsteady eccentric fixation. But again, because of very unsteady fixation and lack of a distinct foveal reflex, this finding was simply an estimate, although nonfoveal fixation was clearly present. Haidinger's brush and afterimage transfer test indicated normal retinal correspondence. No ocular or neurological disease was detected. The patient indicated that he would be willing to undergo therapy in order to improve vision in his amblyopic eye.

The initial diagnostic tests and the first phase of orthoptics were conducted prior to the authors' involvement in the case. Orthoptics procedures included occlusion (inverse occlusion during the day and direct occlusion in the evening); placing and maintaining the Haidinger's brush or transferred afterimage on small acuity targets; eye-hand coordination activities such as tracing and tracking objects; accommodation "jump focus" exercises to develop facility of accommodation; and at-home and in-office pleoptics according to Bangerter's method. During this 6-month training period, eccentric fixation decreased to approximately 3 to 5 prism diopters temporal, and visual acuity was at times as high as 20/50. However, neither measure was stable. The second phase of orthoptics training in which new clinicians began their rotation lasted 10 months. The authors became involved in eye movement testing, as well as some phases of clinical testing and training, during the last 8 months of the second phase. The initial comprehensive examination in the second phase showed no change in refraction and absence of strabismus as judged by repeated cover tests. Eccentric fixation was 2 prism diopters temporal as measured by Haidinger's brush, afterimage transfer, and visuoscopy. Visual acuity was 20/200. Orthoptics training procedures similar to those used in the first phase were instituted, with the following additional techniques or modifications introduced at the indicated improved acuity levels: antisuppression training (20/90), fusion training (20/70), direct occlusion (20/50), and saccadic and pursuit tracking (20/25). A variety of therapeutic measures were implemented in order to increase the probability of a functional cure. It is difficult to determine whether all procedures were of equal benefit

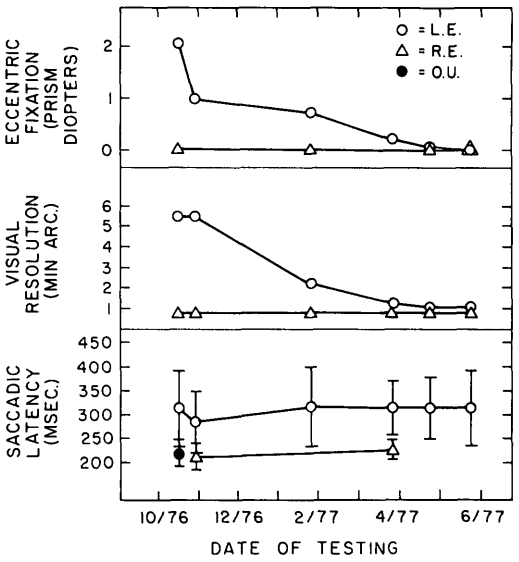

Fig. 1. Changes in eccentric fixation, visual resolution, and saccadic latency for both eyes during last 8 months of orthoptics therapy. Note centralization of fixation and normalization of visual resolution, but maintenance of increased saccadic latencies in left amblyopic eye. Plotted are mean (and standard deviations for saccadic latency) of measures for each test session.

to the patient, and future clinical research in this direction will aid the clinician in optimizing his amblyopia treatment plan. Stereopsis was less than 800 sec arc early in the first phase of training, but it improved to approximately 60 sec arc once 20/20 visual acuity was attained. Eye movements were recorded on six separate occasions during this second phase of training. Two months after 20/20 acuity was attained, the patient moved to another state and was therefore not available for follow-up testing.

Results

Saccadic eye movements. One of the most consistent findings of our study was the persistence of increased saccadic latencies in the amblyopic eye (Fig. 1). As fixation centralized and visual acuity normalized, saccadic latency remained approximately 100 msec longer for monocular tracking with the amblyopic eye than for either binocular tracking or monocular tracking with the dominant eye. Saccadic latency did not vary as a function of target eccentricity for the relatively small range of values tested. Static overshooting[5] (the primary saccade was larger than required, thus necessitating a secondary corrective saccade occurring ap-

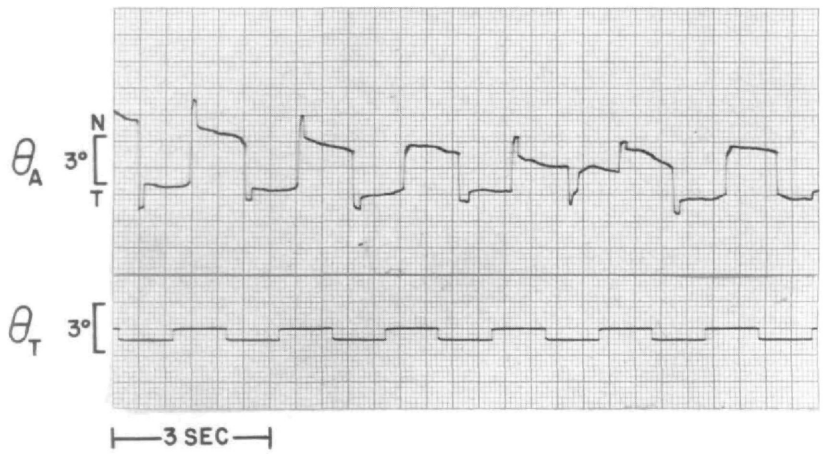

Fig. 2. Monocular saccadic tracking amblyopic eye (20/20). Target amplitude and frequency, 0.6 deg and 0.5 Hz, respectively. Note frequent large static overshoots, as well as increased fixation (amplitude) levels following the overshoots. Intersaccadic intervals for overshoots generally ranged from 100 to 200 msec. Temporal drift more prominent than nasal drift during brief fixation periods. Θ_A, Eye position amblyopic eye; Θ_T, target position; *T*, templeward movements; *N*, nasalward movements.

proximately 100 to 200 msec later) and glissadic undershooting[5] (slow "drifting" eye movements with return velocities ranging from 2 to 20 deg/sec and resulting from pulse-step mismatches in the saccadic controller signals) were also observed. Large static overshoots (0.8 to 2.4 deg in amplitude) were found at times throughout the course of treatment for both random and predictable step tracking, and this was particularly pronounced for tracking small (0.6 deg), predictable (0.5 Hz) step displacements (Fig. 2). Saccadic gain greater than unity for small-amplitude step tracking was evident from the large static overshooting. Intersaccadic intervals for corrective saccades of the overshoots ranged from 100 to 200 msec and thus did not exhibit delays. Overshoot amplitude was independent of target amplitude. Also present were increased fixation (amplitude) levels following the overshoots. Marked glissadic undershooting was no longer a frequent finding once 20/20 visual acuity was attained; it occurred approximately 30% of the time at 20/110 visual acuity but less than 5% of the time at 20/20. Tracking of random-pulse inputs and small-amplitude step displacements was also tested. The amblyopic eye responded to pulses as short as 80 msec in duration and, at times, to random step displacements as small as 0.4 deg in amplitude, at every test session. The amblyopic eye responded, at times, with multiple saccades to combinations of pulse and step stimuli. After the initial delayed saccade (~300 msec delay), remaining saccades in such a train of multiple saccades often had normal initiation times (~150 to 200 msec). The normal eye would also, at times, respond with multiple saccades but without the initial delay to combinations of pulse and step stimuli. This indicates that the motor control aspects of generation and initiation of saccades were not slowed or prolonged; only the sensory processes involved in initiation of the first saccade of a train of multiple saccades was prolonged or delayed consequent to the amblyopic defect.[6–8] Two other aspects of saccadic eye movements that support normal motor mechanisms in this patient were normal saccadic durations for monocular tracking with the amblyopic eye[6–8] and normal saccadic latencies for binocular tracking and monocular tracking with the dominant eye.[6–8] Saccadic accuracy was normal for binocular tracking and monocular tracking with the dominant eye. During binocular tracking, dynamic violations of Hering's law, as commonly found in normal subjects,[9] were found in our amblyopic patient at each test session.

Fixational eye movements. Fig. 3 shows changes in presence of abnormal drift (number of 1 sec intervals in which increased drift was found divided by total number of seconds fixation was tested), average maximum drift velocity and amplitude, eccentric fixation, and visual resolution during the course of orthoptics treatment. In general, drift characteristics tended to normalize, and

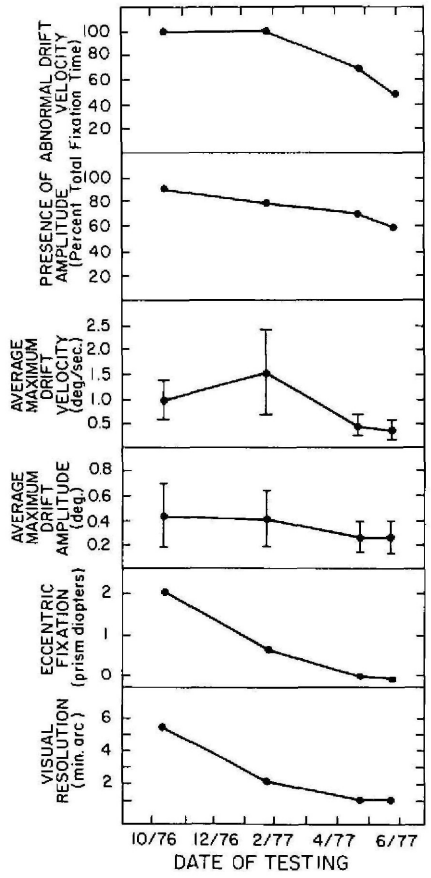

Fig. 3. Changes (for left eye) in presence of abnormal drift velocity and amplitude, average maximum drift velocity and amplitude (with standard deviations), eccentric fixation, and visual resolution during the last 8 months of orthoptics therapy. All show clear normalizing trends.

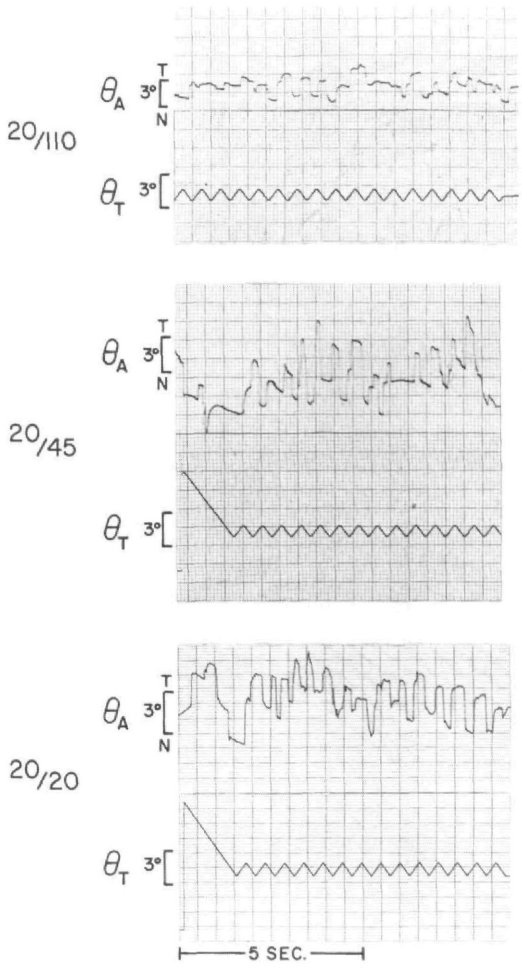

Fig. 4. Monocular pursuit with amblyopic eye as a function of visual acuity level. In each pair of records, top trace is eye position, and bottom trace is stimulus (1.0 deg amplitude, 3.75 deg/sec velocity, 1.88 Hz). Note persistence of abnormal saccadic substitution in spite of acuity improvement; this tracking response was most pronounced at 20/45 level. Symbols same as in Fig. 2.

periods of steady fixation became more frequent as visual acuity and eccentric fixation normalized. The increase in average maximum drift velocity, as well as its increased variability, at 20/45 acuity is of interest; one might speculate that when certain relatively fixed patterns of eccentric fixation are disrupted, control of drift velocity is adversely affected until a new region of fixation is established. Not displayed in Fig. 3 are the maximum drift values found at each session. Maximum peak-to-peak drift amplitude was 1.7, 3.4, 2.2, and 1.0 deg, and maximum drift velocity was 1.5, 3.3, and 1.0, and 0.7 deg/sec at visual acuity levels of 20/110, 20/45, 20/20 (first session), and 20/20 (second session), respectively. Evident at all test sessions was the paucity of single, large saccades and saccadic intrusions during fixation; an intrusion consists of a pair of saccades of approximately equal amplitude but of opposite direction that occur irregularly during fixation at a rate of about one per second with intersaccadic intervals ranging from 150 to 500 msec. These intrusions, commonly found in strabismic patients,[6a] result in little *net* change in eye position. Rather, as found in amblyopes without strabismus,[6] drift was the prominent feature of the records. Drift characteristics were similar for all five amplitudes of horizontal gaze tested. Binocular fixation and monocular fixation with the dominant eye were

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within normal limits (<12 min arc drift amplitude, <20 min arc/sec drift velocity).

Pursuit movements. Two pursuit abnormalities, abnormal saccadic substitution and low pursuit gain, were observed during the course of treatment. Abnormal saccadic substitution[6b] during pursuit of small-amplitude targets (1 and 2 deg) was found at each test session; that is, saccades having amplitudes generally two to five times larger than the target amplitude were used to track the target, rather than smooth movements. Representative examples of abnormal saccadic substitution recorded at three acuity levels for the same small-amplitude stimulus (1 deg, 3.75 deg/sec) are shown in Fig. 4. Large saccades, with little evidence of smooth movements, are prominent in each trace. Pursuit gain for tracking of targets having larger amplitudes (4 and 8 deg) was also of interest. When visual acuity was 20/110, long duration (up to 1 sec), low gain (0.1 to 0.2 range, 0.14 mean value) pursuit, followed by large saccades correcting for position errors, was observed. Low, variable pursuit gain (0.0 to 0.8 range, 0.45 mean value) continued at the 20/45 acuity level. However, once visual acuity reached 20/25, pursuit gain was generally higher and less variable (0.5 to 0.8 range, 0.60 mean value). Pursuit gain for monocular tracking with the dominant eye generally ranged from 0.7 to 0.95, with a mean value of 0.85 (within normal limits for our laboratory). Conjugate movements were always present during binocular pursuit tracking.

Discussion. To the best of our knowledge, this is the first report in the literature providing quantitative eye movement analysis in an amblyope during an extended period of orthoptics treatment. Although only one patient was studied, we believe that our results and their clinical implications are of interest and importance to those involved in clinical, as well as theoretical, aspects of amblyopia and its treatment.

Several aspects of eye movement control improved in the amblyopic eye during treatment: decrease in drift amplitude, decrease in drift velocity, increase in frequency and duration of steady fixation, and increase in pursuit gain. These findings demonstrate that as amblyopia decreased and fixation became centralized, certain aspects of eye movement control (primarily under the province of the smooth-pursuit system) could be modified by the orthoptics therapy. Furthermore, the findings suggest that the "critical period" for oculomotor plasticity for these aspects of eye movement control in our amblyope extended into adulthood.

However, other aspects of eye movement control in the amblyopic eye remained abnormal throughout treatment: increased saccadic latencies, abnormal saccadic substitution, and static overshooting. The increase in saccadic latencies suggests a processing delay over the central retina in the amblyopic eye involving pathways from the amblyopic eye to centers controlling saccadic initiation, such as the superior colliculus,[8] although involvement of parietal lobe mechanisms underlying directed visual attention remains a distinct possibility.[10] Furthermore, the results represented by Fig. 1 suggest that in amblyopia those neural channels conveying and/or processing spatial resolution information (probably "sustained" channels[11]), as well as those neural elements responsible for eccentric fixation, can be functionally modified in the adult amblyope and require a relatively short course of therapy for recovery (the visual acuity recovery function for our adult amblyope was similar to that measured in a very young amblyope[12]). This is in contrast to those neural channels (probably "transient" channels[11]) involved in saccadic initiation, where recovery in an adult amblyope is either very slow or no longer possible. This is consistent with a recent finding of residual differences in visual-evoked potential peak latencies of greater than 5 msec between the two eyes of former amblyopic patients now having equal vision in each eye, thus suggesting that electrophysiological changes remain despite a clinical cure of the amblyopia.[13] The abnormal saccadic substitution response found for tracking a small target moving smoothly over the retina suggests a defect in direction sense[6b] (i.e., difficulty in estimating small angular changes in target location; in this case, overestimation seems to be the rule), and this may be related to the increased saccadic gain and increased fixation (amplitude) levels that were prominent during small-amplitude step tracking (Fig. 2).

A possible complicating factor in this case was the effect of form deprivation during the first 2 days of life. A recent report by Movshon and Dürsteler[14] clearly shows significant shifts in ocular dominance, broadening of cortical receptive field orientation specificity, and reduction in lateral geniculate nucleus cell size with only 1 to 2 days of unilateral eye closure in kittens during the peak of the critical period. Although a critical period for man has not yet been firmly established, the possibility remains that a short period of form deprivation very early in life could have adverse effects on the postnatal development of the human visual system.[15] It is remarkable that

several aspects of oculomotor control, as well as visual acuity and fixation, showed such improvement in spite of numerous indications of an unfavorable prognosis for a functional cure, including initiation of therapy in adulthood, presence of deep amblyopia with eccentric fixation, minimal use of spectacles until high school, and possible effects of form deprivation.

The fixation and tracking abnormalities recorded during the course of therapy would be most difficult to quantitatively assess by standard clinical techniques. Our results suggest that after normalization of visual acuity and centralization of fixation (the hallmarks of cured amblyopia), subtle defects in oculomotor performance may persist, clearly demonstrating that all vision functions in the amblyopic eye do not improve concurrently. Thus it appears that orthoptics therapy for amblyopia should perhaps be continued until visual acuity, fixation, and oculomotor control (as well as other vision functions) are normalized and/or remain stable for a suitable period of time. Objective recording and analysis are essential to quantify the dynamic aspects of eye movements in the amblyopic eye and to monitor the return and persistence of normal function. Moreover, those particular objectively determined eye movement measures that changed and tended to normalize during treatment in our patient, if used in conjunction with the subjectively determined visual acuity measures, could provide the clinician a firmer base upon which to decide when the eye had been satisfactorily treated and therapy could be terminated. Perhaps lack of normalization of the oculomotor system results in some improved or "cured" amblyopic patients reverting to their former condition after termination of therapy. Thus accurate recordings of eye movement control before, during, and after treatment may be a valuable tool in assessing the functional cure of amblyopia.

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REFERENCES

1. Burian, H. M., and von Noorden, G. K.: Binocular Vision and Ocular Motility, St. Louis, 1974, The C. V. Mosby Co., pp. 420-423.
2. Ludlam, W. M.: Orthoptic treatment of strabismus, Am. J. Optom. Arch. Am. Acad. Optom. **38**:369, 1961.
3. Simons, K., Moss, A., and Reinecke, R.: Ocular motility test administration analysis by computer in strabismus and amblyopia evaluation, Comput. Biol. Med. **8**:105, 1978.
4. von Noorden, G. K., and Burian, H. M.: An electro-oculographic study of the behavior of the fixation in amblyopic eyes in light- and dark-adapted state: a preliminary report, Am. J. Ophthalmol. **46**:68, 1958.
5. Bahill, A. T., Clark, M. R., and Stark, L.: Dynamic overshoot in saccadic eye movements is caused by neurological control signal reversals, Exp. Neurol. **48**:107, 1975.
6. Ciuffreda, K. J.: Eye movements in amblyopia and strabismus, Ph.D. dissertation, School of Optometry, University of California, Berkeley, 1977.

6a. Ciuffreda, K. J., Kenyon, R. V., and Stark, L.: Saccadic intrusions in strabismus, Arch. Ophthalmol. (in press).

6b. Ciuffreda, K. J., Kenyon, R. V., and Stark, L.: Abnormal saccadic substitution during small-amplitude pursuit tracking in amblyopic eyes, INVEST. OPHTHALMOL. VISUAL SCI. (in press).

7. Ciuffreda, K. J., Kenyon, R. V., and Stark, L.: Processing delays in amblyopic eyes: evidence from saccadic latencies, Am. J. Optom. Physiol. Opt. **55**:187, 1978.
8. Ciuffreda, K. J., Kenyon, R. V., and Stark, L.: Increased saccadic latencies in amblyopic eyes, INVEST. OPHTHALMOL. VISUAL SCI. **17**:697, 1978.
9. Bahill, A. T., Ciuffreda, K. J., Kenyon, R. V., and Stark, L.: Dynamic and static violations of Hering's law of equal innervation, Am. J. Optom. Physiol. Opt. **53**:786, 1976.
10. Mountcastle, V. B.: Brain mechanisms for directed attention. J. R. Soc. Med. **71**:14, 1978.
11. Ikeda, H., and Wright, M. J.: Is amblyopia due to inappropriate stimulation of the "sustained" pathway during development? Br. J. Ophthalmol. **58**:165, 1974.
12. Mohindra, I.: Early treatment of anisometropic astigmatism and strabismus, Am. J. Optom. Physiol. Opt. **54**:479, 1977.
13. Lawwill, T.: Electrophysiologic aspects of amblyopia, Trans. Am. Acad. Ophthalmol. Otolaryngol. **85**:451, 1978.
14. Movshon, J. A. and Dürsteler, M. R.: Effects of brief periods of unilateral eye closure on the kitten's visual system, J. Neurophysiol. **40**:1255, 1977.
15. Awaya, S., Mikake, Y., Imaizumi, Y., Shiose, Y., Kanda, T., and Komuro, K.: Amblyopia in man, suggestive of stimulus deprivation amblyopia, Jpn. J. Ophthalmol. **17**:69, 1973.